

## *Original Article*

# **The Renin–Angiotensin–Aldosterone System During Haemodialysis With Acetate or Bicarbonate at Different Dialysate Sodium Concentrations**

B. K. Krämer, K. M. Röss, T. M. Ulshöfer and T. Risler

III Department of Internal Medicine, University of Tübingen, Tübingen, FRG

**Abstract.** The hormones of the renin–angiotensin–aldosterone system were measured during regular haemodialysis with acetate or bicarbonate at dialysate sodium concentrations of 135, 140, 145, and 150 mmol/l. Plasma renin activity and aldosterone concentration were higher during acetate haemodialysis than during bicarbonate haemodialysis. At lower dialysate sodium concentrations, plasma renin activity (acetate dialysis and bicarbonate dialysis) and aldosterone concentration (only acetate dialysis) were higher than they were at higher dialysate sodium concentrations. Plasma renin activity increased during acetate dialysis, but did not change during bicarbonate dialysis. Aldosterone and potassium concentrations were positively correlated. Aldosterone decreased during haemodialysis (increase to predialysis values at the end of haemodialysis (4 h) at lower dialysate sodium concentrations). It is concluded that the renin–angiotensin–aldosterone system is activated more during acetate dialysis than during bicarbonate dialysis. Aldosterone concentrations seem to be related more closely to serum potassium than to renin–angiotensin–aldosterone system and to serum sodium intradiallytically.

**Key words:** Acetate; Bicarbonate; Haemodialysis; High–low sodium dialysis; Renin–angiotensin–aldosterone system.

## **Introduction**

Dialytic intolerance to fluid removal is often a problem in patients during haemodialysis. Either fluid and sodium overload [1–3] or the renin–angiotensin–aldosterone system ('inappropriately high plasma renin activity') are

important in haemodynamic stability and blood pressure regulation in patients with end-stage renal disease. The present study examines the renin–angiotensin–aldosterone system at conditions of water deprivation during regular haemodialysis with different buffers and different dialysate sodium concentrations.

## **Patients and Methods**

Six patients with end-stage renal disease were examined. Clinical data was as follows: chronic haemodialysis for 0.5–7.0 years; aged 17–66 years; three male and three female. Aetiology of the renal failure was three polycystic kidneys, two chronic pyelonephritis, and one hydro-nephrosis. Daily urine output was <100 ml in four patients and <600 ml in the other two; no patient received antihypertensive treatment, none was bilaterally nephrectomised.

The subjects were examined during acetate dialysis at four different dialysate sodium concentrations: 135, 140, 145 and 150 mmol/l. In each case haemodialysis with the same dialysate sodium concentration had been performed 2 days before the haemodialysis entered in this study. Mean volume removal was 2.10 litres during bicarbonate dialysis and 2.15 litres during acetate dialysis. Five of the six patients were examined during bicarbonate dialysis at the same four sodium concentrations.

Blood samples were obtained from the arterial line before dialysis, after 1 h 20 min, after 2 h 40 min, and after 4 h of dialysis (end of haemodialysis). Plasma renin activity, aldosterone concentration, angiotensin II concentration and serum sodium and potassium concentrations were measured. Blood pressure was measured every 20 min by means of a cuff sphygmomanometer.

**Table 1.** Plasma renin activity, angiotensin II and aldosterone: Mean value of measurements during haemodialysis and range of values

	Acetate dialysis (n = 5)	Bicarbonate dialysis (n = 5)	P
Plasma renin activity (ng/ml per h)			
$\bar{x} \pm \text{SEM}$	$2.13 \pm 0.30$	$0.95 \pm 0.13$	<0.0001
range	0.07–9.20	0.07–4.81	
Angiotensin II (pg/ml)			
$\bar{x} \pm \text{SEM}$	$90.7 \pm 10.9$	$28.9 \pm 1.7$	<0.0001
Aldosterone (pg/ml)			
$\bar{x} \pm \text{SEM}$	$515 \pm 67$	$446 \pm 62$	<0.05
range	41–2400	34–2400	

Range of normal values: plasma renin activity (supine position), 0.12–1.59 ng/ml per h; angiotensin II, 5–15 pg/ml; aldosterone (supine position), 10–160 pg/ml

**Table 2.** Diastolic blood pressure, plasma renin activity and aldosterone concentration. Mean values of measurements during dialysis at each sodium concentration in dialysate (cNaD/mmole per l)

Plasma renin activity (ng/ml per h)	Acetate dialysis (n = 6)	Bicarbonate dialysis (n = 5)
cNaD = 135	$2.98 \pm 0.70$	$1.39 \pm 0.37$
140	$2.78 \pm 0.63$	$0.94 \pm 0.24$
145	$2.12 \pm 0.50$	$0.77 \pm 0.20$
150	$1.55 \pm 0.39$	$0.72 \pm 0.20$
Aldosterone (pg/ml)	Acetate dialysis (n = 6)	Bicarbonate dialysis (n = 5)
cNaD = 135	$760 \pm 142$	$546 \pm 129$
140	$653 \pm 136$	$416 \pm 120$
145	$515 \pm 112$	$390 \pm 125$
150	$364 \pm 72$	$430 \pm 127$
Diastolic blood pressure (mmHg)	Acetate dialysis (n = 6)	Bicarbonate dialysis (n = 5)
cNaD = 135	$71.5 \pm 1.2$	$68.5 \pm 1.5$
140	$75.5 \pm 2.1$	$77.0 \pm 2.2$
145	$77.5 \pm 1.7$	$74.0 \pm 2.1$
150	$80.3 \pm 1.7$	$73.4 \pm 1.3$

\* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ .

Haemodialysis was performed with the dialyser C-DAK Artificial Kidney 135scc (CD Medical GmbH, Munich, FRG); 1.4 m<sup>2</sup> surface, saponified cellulose ester, with a dialysate flow of 444 ml/min, and a blood flow of 250 ml/min (Cobe Centry 2000 Rx; Cobe Laboratories GmbH, Kirchheim/München, FRG).

We used dialysate preparations for bicarbonate dialysis (Fa. Fresenius AG, Bad Homburg, FRG; PGS 22+8.4% NaHCO<sub>3</sub>) and for acetate dialysis (Fa. B. Braun, Melsungen, FRG; Y 108). Final dialysate concentrations were: potassium 2.0–2.2 mmol/l; sodium 135, 140, 145, 150 mmol/l; acetate 35–39 mmol/l; bicarbonate 31–35 mmol/l (+3 mmol/l acetate).

Commercially available kits were used to measure plasma renin activity (Fa. Serono Diagnostika GmbH, Freiburg, FRG; Renin MAIA), and angiotensin II and aldosterone (Fa. H. Biermann Medizinische Systeme GmbH, Bad Nauheim, FRG; J-125 Angiotensin II and Coat-A-Count Aldosteron).

Sodium and potassium concentrations were measured with ion-selective electrodes (Nova I, Nova Biomedical GmbH, Darmstadt, FRG). Dialysate sodium and

potassium concentrations were controlled by direct measurements.

Statistical evaluation was performed by means of Student's paired t test, and the coefficient of correlation. *P* values less than 0.05 were considered statistically significant. Data are expressed as mean  $\pm$  SEM. To improve legibility, in Figs 1–3, SEM are not drawn in at all dialysate sodium concentrations.

## Results

Plasma renin activity, angiotensin II and aldosterone concentration were higher during acetate dialysis than during bicarbonate dialysis (Table 1).

Mean values of plasma renin activity, aldosterone concentration and diastolic blood pressure during acetate dialysis and bicarbonate dialysis at different dialysate sodium concentrations are shown in Table 2.

Because of rather large individual differences in plasma renin activity and aldosterone concentration, we examined plasma renin activity and aldosterone in relation to

individual plasma renin activity and aldosterone values predialysis at 135 mmol/l dialysate sodium concentration. During acetate dialysis relative plasma renin activity increased ( $P < 0.01$ ), but during bicarbonate dialysis there was no significant change of relative plasma renin activity (Fig. 1). Relative aldosterone concentration decreased during acetate dialysis at 145 and 150 mmol/l dialysate sodium concentration in comparison to predialysis values ( $P < 0.01$ ). At 135 and 140 mmol/l during acetate dialysis a decrease ( $P < 0.05$  or  $0.01$ ) in relative aldosterone concentration was demonstrated at 1 h 20 min and at 2 h 40 min, but not at 4 h, the end of haemodialysis (Fig. 2).

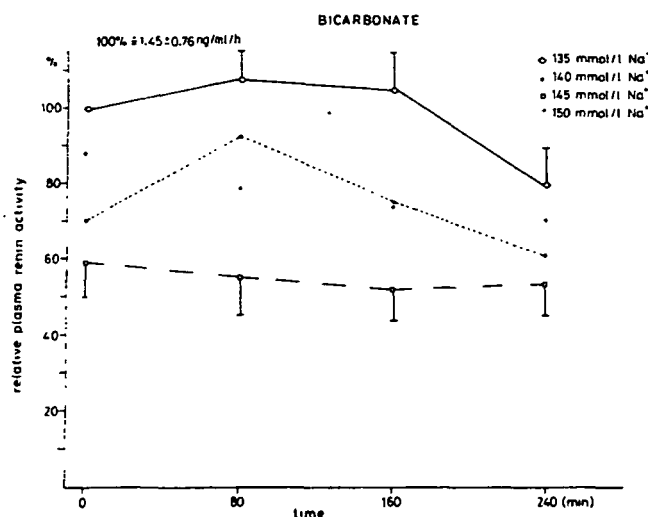


Fig. 1. Relative plasma renin activity during haemodialysis with bicarbonate.

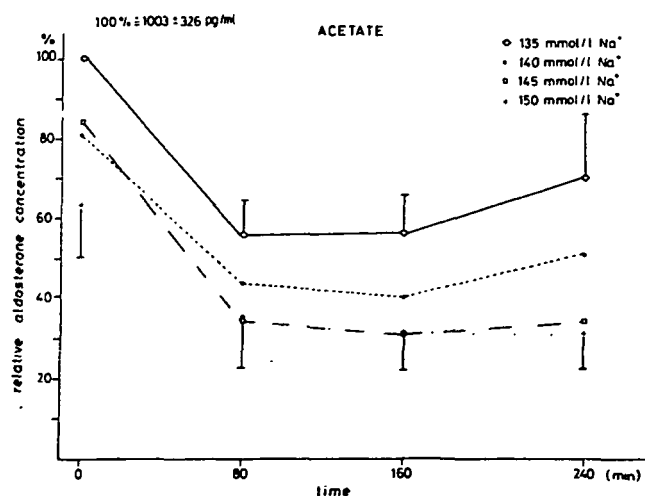


Fig. 2. Relative aldosterone concentration during haemodialysis with acetate.

During bicarbonate dialysis relative aldosterone concentration decreased at 140, 145 and 150 mmol/l dialysate sodium concentration in comparison to predialysis values

( $P < 0.05$  or  $0.01$ ). During bicarbonate dialysis at 135 mmol/l dialysate sodium concentration, relative aldosterone concentration showed a significant decrease at 1 h 20 min and at 2 h 40 min, but not at 4 h (Fig. 3).

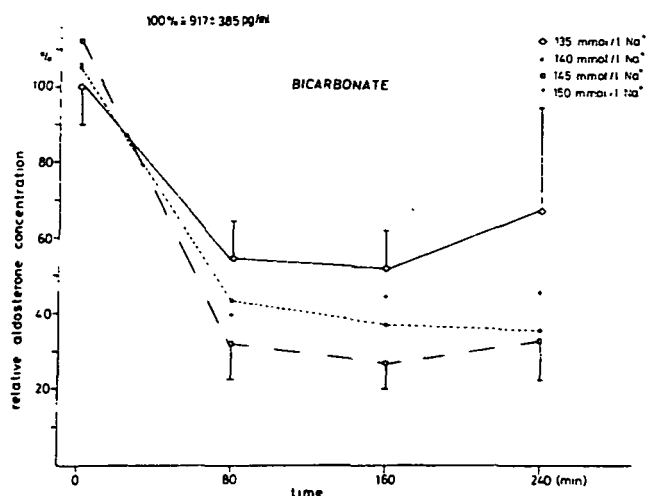


Fig. 3. Relative aldosterone concentration during haemodialysis with bicarbonate.

Serum sodium concentration correlated negatively with relative plasma renin activity values ( $r = -0.45$  in acetate dialysis,  $r = -0.66$  in bicarbonate dialysis;  $P < 0.01$  each). Volume loss correlated positively with the plasma renin activity relative to the predialysis values at each dialysate sodium concentration during acetate dialysis ( $r = 0.44$ ;  $P < 0.01$ ), but not during bicarbonate dialysis.

Serum sodium concentration correlated negatively with relative aldosterone concentration ( $r = -0.38$  (acetate dialysis),  $r = -0.40$  (bicarbonate dialysis);  $P < 0.01$  each).

Serum potassium concentration decreased during haemodialysis ( $P < 0.01$ ) and correlated positively with aldosterone and relative aldosterone concentration ( $r = 0.45$  (acetate dialysis),  $r = 0.55$  (bicarbonate dialysis);  $P < 0.01$  each) (Fig. 4). Plasma renin activity correlated positively with aldosterone concentration ( $r = 0.72$  (acetate dialysis),  $r = 0.70$  (bicarbonate dialysis),  $P < 0.01$  each).

Diastolic blood pressure showed a slight but not significant tendency to decrease during acetate dialysis and bicarbonate dialysis. During acetate dialysis low diastolic blood pressure is demonstrated at low dialysate sodium concentrations ( $RR_{135}^{**} < RR_{140}^{n.s.} < RR_{145}^{*} < RR_{150}$ ;  $*P < 0.05$ ,  $**P < 0.01$ ). During bicarbonate dialysis, diastolic blood pressure at 135 mmol/l dialysate sodium concentration was significantly less than at higher dialysate sodium concentrations, but no further increase in blood pressure with increasing dialysate sodium concentration was demonstrated.

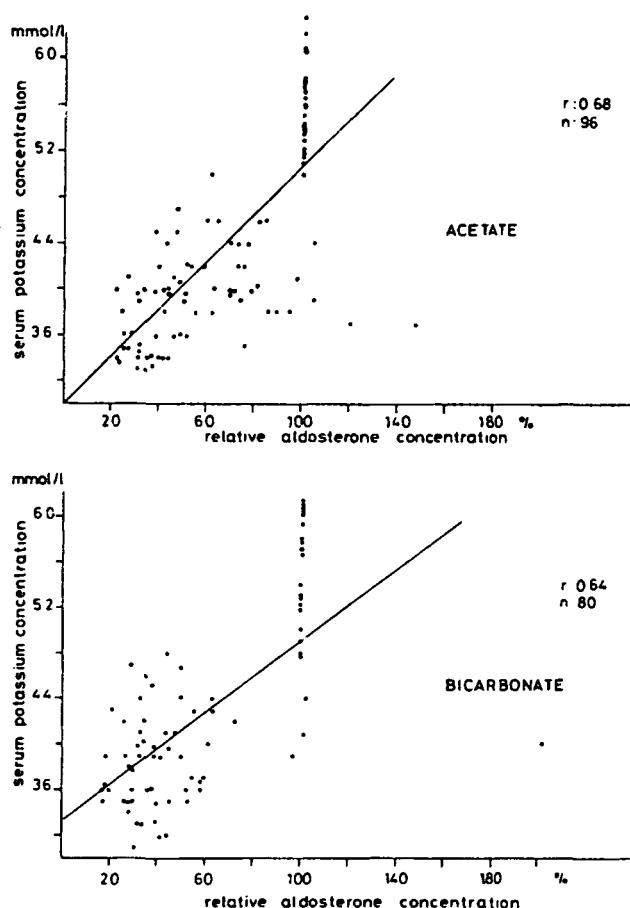


Fig. 4. Relative aldosterone concentration (predialytic value = 100%) and serum potassium concentration during haemodialysis with acetate and bicarbonate.

Heart rate was significantly higher during acetate dialysis than during bicarbonate dialysis ( $P < 0.001$ ); it increased, but not significantly, during acetate dialysis (approximately 10 per min), and was unchanged during bicarbonate dialysis.

Serum sodium concentration increased during bicarbonate dialysis at 150 mmol/l dialysate sodium concentration ( $P < 0.05$ ), and decreased during acetate dialysis at 135 mmol/l dialysate sodium concentration ( $P < 0.05$ ); at other dialysate sodium concentrations no significant changes in serum sodium concentration were demonstrated.

## Discussion

Several studies show an elevated plasma renin activity in patients with end-stage renal disease [4–7], although others do not confirm these results [8–9]. Van Stone et al [10] examined six patients during haemodialysis with a dialysate sodium concentration of 131, 142 and 150 mmol/l, and did not find significant differences between the plasma renin activity at the different sodium

concentrations. Regarding patients with kidneys remaining in situ, plasma renin activity was shown to be higher postdialysis than predialysis [5–9, 11, 12]. Van Stone et al [10, 13] confirmed these results in six patients (including one nephrectomised) for all examined dialysate sodium concentrations. Tuma et al [14] and Juncos et al [9] demonstrated a fall in plasma renin activity in nephrectomised patients during haemodialysis.

In some of the above-mentioned studies haemodialysis was performed using acetate as buffer [7, 10, 11, 13, 14], but the buffer employed was not stated in the other studies [4–6, 8, 9, 12]. Thus, our results confirm these reports regarding the increase of plasma renin activity during acetate dialysis in non-nephrectomised patients. Rapid changes in fluid and sodium/potassium content are able to cause high renin release. Juncos et al [9] suggested an increase in plasma renin activity caused by ultrafiltration. The inverse correlation of relative plasma renin activity with serum sodium concentration demonstrated that a low sodium concentration is followed by high plasma renin activity, suggesting that a decrease in sodium concentration (accompanied by decrease of blood volume) stimulates renin release. However, in that study, a decrease in serum sodium concentration was only examined during haemodialysis with low sodium concentrations in the dialysate.

The present results show that serum sodium concentration is not the main factor stimulating renin release during acetate dialysis, because the increase of plasma renin activity during acetate dialysis at a dialysate sodium concentration of 145 and 150 mmol/l occurred in spite of constant serum sodium concentrations. Changes in blood volume are therefore supposed to be more important for renin release.

The role of plasma renin activity is different during acetate dialysis compared to bicarbonate dialysis, there is an increase of plasma renin activity during acetate dialysis but no change during bicarbonate dialysis. This might be due to several side-effects of acetate when used as a dialysate buffer:

- Acetate has a known vasodilating effect [26–31].
- During bicarbonate dialysis blood volume is preserved better than during acetate dialysis, plasma refilling rate is higher, and plasma refilling was said to take place earlier during the haemodialysis procedure [15].
- In addition, acetate possibly has a depressant effect on myocardial function [26, 27, 29, 32, 33], but not all authors support this view [31, 34–36].

Secondary to those mechanisms the renin–angiotensin–aldosterone system is stimulated more during acetate dialysis than during bicarbonate dialysis, with higher values of plasma renin activity, aldosterone concen-

tration, angiotensin II and different dialysate sodium concentrations influencing those levels more when acetate is used as a buffer.

No information concerning the behaviour of the renin-angiotensin-aldosterone system during bicarbonate dialysis, especially in contrast to acetate dialysis, is available in the literature.

Most authors have found elevated aldosterone concentrations in patients with end-stage renal disease [6, 8, 11, 16]. Bonomini et al [4] found an increase in aldosterone concentration with loss of kidney function, but aldosterone concentration remained in the normal range (no patients with end-stage renal disease on chronic haemodialysis were examined). A decrease of aldosterone concentration during haemodialysis was demonstrated by several authors [6, 8, 17, 18, 20].

At different dialysate sodium concentrations, Van Stone et al [10] found a decrease of aldosterone concentration during haemodialysis without weight loss; also at a dialysate sodium concentration of 152 mmol/l with a 2-kg weight loss. During haemodialysis at 131 and 142 mmol/l sodium concentration and 2-kg weight loss, however, an increase in aldosterone concentration was reported.

Henrich et al [20] demonstrated a decrease in aldosterone concentration despite an increase in plasma renin activity during regular haemodialysis, whereas when potassium concentration was kept constant, an increase of plasma renin activity as well as of aldosterone concentration was demonstrated. The authors concluded that the loss of the stimulus of a high serum potassium concentration, with potassium decreasing during haemodialysis, would override the influence of fluid depletion and increased plasma renin activity on aldosterone secretion. Weidmann and Maxwell [23] reported similar results.

In the present study a decrease of aldosterone concentration both during acetate dialysis and bicarbonate dialysis was shown, confirming the results of Henrich et al [20] and Weidmann and Maxwell [23].

Thus a decrease of aldosterone concentration, being an index/measure of aldosterone release because of aldosterone's short half-life of 29.7 min in plasma [27], is thought to be caused mainly by the decrease in serum potassium concentration.

At lower sodium concentrations in the dialysate, however, aldosterone concentration reached predialysis concentrations again at the end of haemodialysis. At the end of haemodialysis, high potassium concentrations, as a stimulus to aldosterone release, no longer exist. Therefore the release of aldosterone at the end of haemodialysis has to be stimulated by other factors such as plasma renin activity. Plasma renin activity has been shown to be highest at low dialysate sodium concentrations and to increase during acetate dialysis. Thus plasma renin activity is thought to be one cause of the increase of

aldosterone concentration at the end of low-sodium haemodialysis with weight loss, the increase of aldosterone concentration being more marked during acetate dialysis than during bicarbonate dialysis.

Another possible reason for the intradialytic decrease of aldosterone concentration could be the loss of aldosterone across the dialyser. Aldosterone is a mid-sized molecule with a molecular weight of about 380 U, and is dialysable. However, according to Iitake et al [25] the fall in postdialysis aldosterone concentration cannot be explained by the loss of plasma aldosterone into the dialysate.

High levels of plasma renin activity and angiotensin II during acetate dialysis contribute to stabilisation of blood pressure. Blood pressure is not lower during acetate dialysis compared to bicarbonate dialysis in spite of the vasodilatory effect of acetate and the slower plasma refilling. However, diastolic blood pressure is thought to depend more on dialysate sodium concentration during acetate dialysis than during bicarbonate dialysis.

In conclusion, the renin-angiotensin-aldosterone system is activated more during acetate dialysis than during bicarbonate dialysis, and more at lower dialysate sodium concentrations than at higher concentrations. Higher activation of the renin-angiotensin-aldosterone system during acetate dialysis is thought to be secondary to the vasodilatory effect of acetate and the slower refilling of plasma volume. Either the renin-angiotensin-aldosterone system or serum potassium concentration stimulate aldosterone release, and these effects are modulated by other mechanisms such as sensitisation of the zona glomerulosa by sodium depletion [21, 22].

Aldosterone concentration seems to be related more closely to serum potassium concentrations than to the renin-angiotensin-aldosterone system and serum sodium concentrations intradialytically.

## References

1. Blumberg A, Nelp WB, Hegstrom RM, Scribner BH. Extracellular volume in patients with chronic renal disease treated for hypertension by sodium restriction. *Lancet* 1967; 2: 69-73
2. Kornerup HJ. The influence of different sodium loads on renin release in hypertensive and normotensive states of chronic renal failure. *Scand J Clin Lab Invest* 1978; 38: 147-153
3. Kornerup HJ. Pathophysiological and clinical aspects concerning the renin-angiotensin system and the body sodium content in renal and post-renal-transplant hypertension in humans. *Scand J Urol Nephrol [Suppl]* 1980; 56: 1-58
4. Bonomini V, Orsoni G, Stefoni S, Vangelista A. Hormonal changes in uremia. *Clin Nephrol* 1979; 11: 275-280
5. Craswell PW. Renin secretory capacity of end-stage kidney in patients on regular hemodialysis. *Dial Transplant* 1982; 11: 492-495
6. Hammer M, Dawids S, Sørensen MB, Christensen O, Ølgaard K. Hormonal response to volume depletion in non-nephrectomised patients on regular haemodialysis. *Proc Eur Dial Transplant Assoc* 1979; 16: 252-260
7. Imai Y, Abe K, Otsuka Y et al. Blood pressure regulation in chronic hypotensive and hypertensive patients with chronic renal failure. *Jpn Circ J* 1981; 45: 303-314

8. Krause JZ, Matarese RA, Zabetakis PM, Michelis MF. Reversibility of hyporeninemia and hypoaldosteronemia in chronic hemodialysis patients by correction of fluid excess. *J Lab Clin Med* 1980; 96: 734-742
9. Juncos LI, Fuller TJ, Cade JR. Effects of ultrafiltration on peripheral plasma renin activity and prostaglandin concentration in hemodialysis patients with and without bilateral nephrectomy. *J Lab Clin Med* 1977; 90: 904-913
10. Van Stone JC, Bauer J, Carey J. The effect of dialysate sodium concentration on body fluid compartment volume, plasma renin activity and plasma aldosterone concentration in chronic hemodialysis patients. *Am J Kidney Dis* 1982; 2: 58-64
11. Ratge D, Augustin R, Wisser H. Catecholamines, renin, aldosterone and arterial pressure in patients on chronic hemodialysis treatment. *Int J Artif Organs* 1983; 6: 255-260
12. Vendemia F, Morganti A, Scalia A, Sala C, Turolo L, D'Amico G. Effects of haemodialysis on active and inactive renin in nephric and anephric patients. *Proc Eur Dial Transplant Assoc* 1983; 20: 514-519
13. Van Stone JC, Bauer J, Carey J. The effect of dialysate sodium concentration on body fluid distribution during hemodialysis. *Trans Am Soc Artif Intern Organs* 1980; 26: 383-386
14. Tuma J, Záruba K, Studer A et al. Regulation der Plasmaaldosteronkonzentration bei nierenlosen Patienten. *Klin Wochenschr* 1981; 59: 27-34
15. Hsu CH, Swartz RD, Sommermeyer MG, Raj A. Bicarbonate hemodialysis: Influence on plasma refilling and hemodynamic stability. *Nephron* 1984; 38: 202-208
16. Coruzzi P, Novarini A, Musiari L, Rossi E, Borghetti A. Effects of 'central hypervolemia' by water immersion on renin-aldosterone system and ACTH-Cortisol axis in hemodialyzed patients. *Nephron* 1984; 36: 238-241
17. Hory B, Henriët MT. Etude des variations de l'aldostéronémie, de l'activité rénine plasmatique et des taux sériques de l'enzyme de conversion pendant la dialyse au bicarbonate dans l'insuffisance rénale chronique. *Nephrologie* 1983; 4: 229-230
18. Ølgaard K, Madsen S. Regulation of plasma aldosterone in anephric and non-nephrectomized patients during hemodialysis treatment. *Acta Med Scand* 1977; 201: 457-462
19. Farinelli A, Squerzanti R, De Paoli Vitali E et al. Response of plasma aldosterone to sequential ultrafiltration, dialysis and conventional hemodialysis. *Nephron* 1980; 26: 274-279
20. Henrich WL, Katz FH, Molinoff PB, Schrier RW. Competitive effects of hypokalemia and volume depletion on plasma renin activity, aldosterone and catecholamine concentrations in hemodialysis patients. *Kidney Int* 1977; 12: 279-284
21. Balla T, Nagy K, Tarján É, Renczes G, Spät A. Effect of reduced extracellular sodium concentration on the function of adrenal zona glomerulosa: Studies in conscious rats. *J Endocrinol* 1981; 89: 411-416
22. Blair-West JR, Coghlan JP, Cran E, Denton DA, Funder JW, Scoggins BA. Increased aldosterone secretion during sodium depletion with inhibition of renin release. *Am J Physiol* 1973; 224: 1409-1414
23. Weidmann P, Maxwell MH. The renin-angiotensin aldosterone system in terminal renal failure. *Kidney Int* 1975; 8 [Suppl] 5: S219-S234
24. Oelkers W, Brown JJ, Fraser R, Lever AF, Morton JJ, Robertson JIS. Sensitization of the adrenal cortex to angiotensin II in sodium-deplete man. *Circulation Res* 1974; 34: 69-77
25. Iitake K, Kimura T, Matsui K et al. Effect of haemodialysis on plasma ADH levels, plasma renin activity and plasma aldosterone levels in patients with end-stage renal disease. *Acta Endocrinol (Copenh)* 1985; 110: 207-213
26. Aizawa Y, Ohmori T, Imai K, Nara Y, Matsuoka M, Hirasawa Y. Depressant action of acetate upon the human cardiovascular system. *Clin Nephrol* 1977; 8: 477-480
27. Baldamus CA, Ernst W, Frei U, Koch KM. Sympathetic and hemodynamic response to volume removal during different forms of renal replacement therapy. *Nephron* 1982; 31: 324-332
28. Henderson LW. Symptomatic hypotension during hemodialysis. *Kidney Int* 1980; 17: 571-576
29. Kirkendol PL, Devia CJ, Bower JD, Holbert RD. A comparison of the cardiovascular effects of sodium acetate, sodium bicarbonate and other potential sources of fixed base in hemodialysate solutions. *Trans Am Soc Artif Intern Organs* 1977; 23: 399-404
30. Kirkendol PL, Pearson JE, Bower JD, Holbert RD. Myocardial depressant effects of sodium acetate. *Cardiovasc Res* 1978; 12: 127-133
31. Wehle B, Asaba H, Castenfors J, Gunnarsson B, Bergström J. Influence of dialysate composition on cardiovascular function in isovolaemic haemodialysis. *Proc Eur Dial Transplant Assoc* 1981; 18: 153-159
32. Iseki K, Onayama K, Maeda T et al. Comparison of hemodynamics induced by conventional acetate hemodialysis, bicarbonate hemodialysis and ultrafiltration. *Clin Nephrol* 1980; 14: 294-298
33. Liomin E, Schneider H, Streicher E. Hämodynamik bei Hämodialyse, Hämofiltration und Hämodiafiltration Bicarbonat- vs Acetatpufferung. *Klin Wochenschr* 1984; 62: 911-919
34. Chen TS, Friedman HS, Smith AJ, Del Monte ML. Hemodynamic changes during hemodialysis: role of dialysate. *Clin Nephrol* 1983; 20: 190-196
35. Schick EC Jr, Idelson BA, Liang C, Redline RC, Bernard DB. Comparison of the hemodynamic response to hemodialysis with acetate or bicarbonate. *Trans Am Soc Artif Intern Organs* 1983; 29: 25-28
36. Mehta BR, Fischer D, Ahmad M, Dubose TD Jr. Effects of acetate and bicarbonate hemodialysis on cardiac function in chronic dialysis patients. *Kidney Int* 1983; 24: 782-787